the next question to be solved is: how does the nucleus specify the 'mid1 domain' at the cell cortex? One possibility is that nuclear export of mid1p, together with diffusion, targets mid1p to the cell cortex overlying the nucleus. However mid1p associates with the cortex, the question is how its cortical localization is maintained at the cell equator.

Double septin rings assemble at the division site in both fission and budding yeast and, at least in the latter organism, they function as diffusion barriers to confine cytokinesis factors to the division site [11]. But double septin rings form only during anaphase in *S. pombe*, which is too late to influence mid1p distribution. Alternatively, a physical link between the nucleus and the cortex may control mid1p localization. A possible candidate is the endoplasmic reticulum (ER), which is formed by membranes extending from the nuclear envelope to the entire cell cortex [12]. Interestingly, recent studies show that, in budding yeast, this organelle is highly compartmentalized [13], indicating the existence of ER diffusion barriers in this, and perhaps other, organisms. Whether mid1p distribution is dependent on ER membranes or other, as yet unidentified structures, is an issue that remains to be addressed.

Beyond advancing our understanding of cleavage plane specification, these studies [5,6] also highlight the role of microtubule networks in maintaining subcellular organization. Many processes involving directed nuclear movements depend on microtubule pulling, as opposed to pushing. For example, migration of the male pronucleus towards the center of *Caenorhabditis elegans* eggs depends on the combined action of microtubules and dynein motors [14], as does nuclear migration into the bud in budding yeast [15]. On the other hand, microtubule-based pushing of an organelle could be the best solution to the problem of finding the geometrical center in a confined space. As has been noted, precise regulation of microtubule dynamics is essential even in this simple case, since microtubules that are too stable would bend around the cell ends resulting in mispositioning of the nucleus [16]. The use of micromanipulation techniques in cells defective in specific aspects of microtubule dynamics promises rapid advances in our understanding of how cells monitor their spatial organization.

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DOI: 10.1016/j.cub.2005.07.040

Ecology: Linking Species Diversity and Genetic Diversity

Although there is a great deal of interest in the biological diversity of species and of genes, it is only recently that researchers have begun to investigate the processes that exert parallel influences on these different levels of diversity.

Anne E. Magurran

Population geneticists and community ecologists often ask similar sorts of questions. Both sets of scientists are interested in measuring, and understanding, the number and distribution of biological variants found in nature. Indeed the conceptual links between the disciplines are recognized in the most widely used definition of biological diversity, devised by the Convention on Biological Diversity, which refers directly to 'variability among living organisms' and stresses that this includes diversity within species as well as between species [1]. But despite striking parallels in approach there have been few attempts to examine these levels of diversity simultaneously.

Figure 1. The grand trillium, or wake robin, *Trillium grandiflorum*. Photograph by Mark Vellend.

Fortunately, researchers have now begun to explore the correlations between genetic and species diversity and to unravel the processes that underpin this relationship. The most recent contribution to this important field is provided by Mark Vellend [2], whose new paper begins to map out the theory that links these two levels of diversity.

Darwin [3] drew attention to an association between the number of varieties within a species and the number of species found in a genus. Antonovics [4] asserted that the forces maintaining species diversity and genetic diversity are similar. Hubbell's [5] influential neutral theory of biogeography and biodiversity makes explicit connections between the rate at which species are formed and the relative abundance of species in ecological communities.

It seems curious, then, that so few scientists have tried to tackle these two organisational levels of biodiversity concurrently. This is probably a consequence of the burgeoning literature, which makes it hard for researchers to keep abreast of recent advances in anything but their own specialty, and the increasingly fine-scale niche separation between the various disciplines in biology. But it is also an opportunity lost. For example, ecologists have long appreciated the need for rarefaction when comparing the diversity of samples of different sizes [6]. As species richness scales in a non-linear fashion with sample size, with area, and with the time interval over which the information is collected, reporting diversity data as some proportion of sampling effort, or even using ANCOVA to provide a correction factor, introduces considerable bias.

Worse still is the use of unstandardised data to compare localities when sampling intensity has been markedly different, as this reveals more about the sampling methodologies than the intrinsic diversity of the sites. Rarefaction is a re-sampling technique that reduces all data sets to the size of the smallest one, and thus enables a fair comparison to be made [6–8].

Exactly the same problem confronts molecular biologists wishing to compare the genetic diversity of taxa, or detect bottlenecks, or decide conservation priorities. It is only recently, however, that the implications of the relationship between allelic richness and sample size have been appreciated [9,10], and so far genetic analyses have not taken full advantage of the insights gleaned over the years by ecologists.

In light of the parallel evolution of these two fields, it is pleasing to see new work that formally builds bridges between them. Vellend [11] was interested in the loss of both genetic and species diversity in secondary forests relative to primary forests in the northeastern USA. He found that the genetic variation in an emblematic forest plant, the wake robin or grand trillium, *Trillium grandiflorum* (Figure 1), a member of the lily family, was diminished in forests growing on abandoned agricultural land. This result was indicative of genetic drift in small populations. The species diversity of these forests was also lower. Moreover, there was convergence on a set of common species, an effect also seen in impacted freshwater fish assemblages in Trinidad [8]. Land-use history, combined with variation in population and community size, drove the correlation between genetic and species diversity.

These findings prompted Vellend to turn to theory. His latest paper [2] uses simulation models to disentangle the interacting influences of area, immigration rate and environmental heterogeneity on the correlated diversity of genes and species. Most simulations lead to positive and sometimes strong correlations. There are, however, interesting nuances in this pattern. For example, environmental heterogeneity leads to changes in population size that can in turn affect genetic diversity. Another intriguing finding is that the correlation between genetic and species diversity weakens when the genetic diversity of rare species is measured. The conclusion from both the theoretical and empirical studies is that negative species and genetic correlations are theoretically unlikely, and rare in nature.

This work clearly demonstrates the intimate connections between population genetics and community ecology. These disciplines not only tackle similar questions but also examine forms of diversity that are shaped by the same factors. But the extent to which the diversity of one level of biological organization can be

predicted from the other remains uncertain. Much depends on the target species in which genetic diversity is measured.

The new study also raises questions about model construction. Like Hubbell [5], Vellend [2] opts for a zero sum approach, that is the assumption that there is a set number of individuals in a community and that, once one dies, it is replaced by another individual. It is the identity of the replacement individual — whether it is the same species or not — that determines the structure of the community. This constraint may be reasonable in plant communities, where space is often at a premium, but the notion of saturation is much more questionable in animal communities [12].

As with Hubbell's model, Vellend's study [2] provides a necessarily simplified view of the real world but both approaches render an important service in sharpening our thinking about the processes that underlie patterns of biodiversity. Darwin [3] used the metaphor of an 'entangled bank' to illustrate how the rich diversity of life we see around us has arisen from common laws. The manner in which these laws jointly influence different forms of diversity is finally beginning to be revealed.

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DOI: 10.1016/j.cub.2005.07.041

Swarming Motility: It Better Be Wet

When grown on a soft agar surface in a rich medium, cells of *Salmonella typhimurium* elongate, produce extra flagella and move over the surface in a coordinated manner. In mutants with defects in the chemotaxis signaling pathway, the agar plates remain dry and the cells' flagella are short. Recent work shows that the anti-sigma factor controlling late-gene flagellar synthesis is secreted less by flagella when things are dry: the flagellum senses wetness.

Howard C. Berg

Swarming is a specialized form of bacterial motility that develops when cells that swim in broth are grown in a rich medium on the surface of moist agar. The cells become multinucleate, elongate, synthesize large numbers of flagella, secrete surfactants and advance across the surface in coordinated packs [1,2]. Classic work on this was done with species of *Proteus* [3], cells of which swarm even on hard agar, alternating between spreading and non-spreading modes, thus forming terraced colonies [4].

About ten years ago, Harshey and Matsuyama [5] found that *Escherichia coli* and *Salmonella* will swarm if grown on soft agar. *E. coli* K12, which lacks the surface O-antigen, swarms reluctantly in such conditions, preferring Eiken agar, which is more wettable than Bacto agar (Figure 1). Somewhat surprisingly, the chemotactic signaling pathway is required for the transformation to the swarming mode, even for cells that are not otherwise chemotactic [6] (for a recent review of chemotactic signaling, see [7]). The reasons for this requirement remain obscure.

Using gene-expression arrays on developing swarms, Wang *et al*. [8] found that only the 'late' flagellar genes are up-regulated in swarming cells — these include the gene that encodes flagellin, *fliC*, the genes that encode motor force-generating elements, *motA* and *motB*, and the genes that encode other components of the chemotaxis signaling pathway. The rest of the flagellar regulon is not affected. (Genes of the type III secretion pathway associated with *Salmonella*'s pathogenicity island SPI-1 also are up-regulated, so there is a link between the synthesis of flagella and of virulence factors, but this will not be discussed further here.)

Wang *et al*. [9] have now found that late genes also are down-regulated when chemotaxis mutants are transferred from broth to swarm plates, whether these mutants are smooth swimming, such as *cheY* or *cheA*, or tumbly, such as *cheZ* or *cheB*. The phenotype is intriguing: these non-swarming cells have fewer and shorter flagella than wild-type cells, and their colonies are relatively dry. This last observation led Wang *et al*. [9] to try to rescue the swarming phenotype by spritzing water on the dry colonies (using a mist that delivered about 3 μl of water per cm^2 agar surface over a period of about 90 seconds). This technique succeeded admirably: cells in wet colonies swarmed normally.

Now, it is known that late-gene expression, promoted by the